

Culbertson (f.)

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REPORT OF A CASE

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Glycosuric Retinitis.

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REPORT OF A CASE OF GLYCOSURIC RETINITIS.

THE object of this paper is to place the case on record and to demonstrate that there is no typical lesion of the retina characteristic of diabetic retinitis.

History.—November 2, 1877, the mother brought to me her child, Minnie R., æt. 6 years, for treatment. The child was pale, anæmic, bright, fond of going to school and had light eyes and hair, and had been sick with some form of malarial fever, and within the last four weeks had been losing her eye-sight. On examination, I found she could only distinguish daylight with the left eye, and that vision in the right eye = $\frac{6''}{viii}$. On investigation it was found she was passing three pints of slightly acid urine daily, and an examination revealed that its s. g. was 1.025 and that it contained sugar, as shown by Trommer's test. Under the microscope no casts or renal epithelium were observed, and heat and nitric acid attested that there was no albumen in the fluid.

Symptoms.—The eyes were normal in appearance externally, the pupils dilated, and there was no pain in or about the eyeballs. The ophthalmoscope revealed in the left eye, at the outer region of the disc and near the macula, small white spots quite similar to those delineated by Jaeger* in plate xiv., fig.

*Traité des Mal. du Fond de L'Oeil.

66, as occurring in albuminuric retinitis. The outlines of the disc on its outer side were obscured with a woolly veil, its arteries and veins were slightly smaller than normal, and the papilla was abnormally red on the outer side. The retinal arteries were indistinctly seen, the media were clear, and there was no hemorrhagic effusion. In the *right* eye the outlines of the disc were more distinct than in the other eye, as well as the retinal vessels on the disc and over the fundus generally. Still the arteries were slightly indistinct. The same spots were observed at the outer side of the disc as in the left eye, though not so marked. There were no blood extravasations, the media were clear, and there were no pigmental deposits in either eye.

The pathology evidently was retinitis, with plasmic effusion and proliferation of the connective tissue of the retina, with limited fatty degeneration. The cause was the glycosuric process, and probably malaria superinduced the latter. The diagnosis was glycosuric retinitis. The prognosis given was, that we hoped to retain the vision now possessed, and even to improve the sight of the right eye.

The treatment was as follows: She was ordered animal food and aliment free from starch and sugar. It was directed that she be clothed with flannel and rubbed over the surface of the body night and morning with a flesh brush, that she be taken out daily in the air, and that removal from school and reading and use of eyes, be prescribed for the present. She was given elix. iron, quinia and strychnia, and ordered to inhale every night

at bedtime two or three drops of the nitrite of amyl.

November 18.—To continue the same treatment and to use a $+3''$ glass in methodic exercise of the left eye and a $+14''$ for the right eye, as well as the $+8''$ lens. Her vision has improved in the right eye, but not in the left. R.V. = $\frac{12''}{iii}$ with a $+12''$ glass. For constipation, she was given $\frac{1}{2}$ gr. pulv. rhei every night. She does not now get up at night to urinate, and I find no sugar in her urine.

December 22d.—Was given zinci phos. gr. ij., ext. nucis vomicæ gr. ijss., iron by hydrogen $\mathfrak{D}j$, 20 pills, S.—one after meals. To continue the methodic exercise, rubbing, amyl, and laxative pill.

January 9, 1878.—To continue same treatment and add $\frac{1}{2}$ grain of santonine three times daily as a retinal tonic. She was also to bathe about the eyes, brows and temples with laudanum.

January 26th.—To continue exercise and laudanum. To omit the other agents, and take elix. iron, quinine and strychnia, also pot. iod. in comp. syrup of sarsap. (The guaiac in this I believe to be useful and the senna beneficial as a laxative.)

February 18.—To omit all agents and to take strychnia granules, each $\frac{1}{40}$ gr., tr. die, and emulsion of cod liver oil.

March 9.—Ordered elix. iron, quinia and strychnia, and pot. iodide; other agents omitted.

On examining the eyes I found the stroma of the left retina was atrophied at spots, the disc was shallow-cupped, its outlines distinct, irregular and pigmented, and its surface blue-white. The retinal arteries and veins were

much reduced in size. In this eye she had only qualitative perception of light. The iris was still active in this, as well as in the right eye. I could not make out the limits of the macula. The right presented the white spots observed in it during the first examination, which extended nearly to the macula and over a larger area than at first. The retinal vessels were now distinct on the disc and over the fundus, and the macula was normal. The color of the disc was pale, and there were none of the finer vessels, and its margins were slightly irregular. The retina generally did not present the bright and distinct granular aspect, but gave a slightly dull reflex, with a $+12''$ lens $V=\frac{8}{11}''$

In the left eye the disease ended in general atrophy of the retina, the diseased process being mainly located at and about the optic nerve entrance—an optic neuritis, essentially—and in the right eye the atrophy was limited to points of the retina, the macula escaping injury, and the influence of the disease not being so profound in its effects upon the optic nerve.

This case, then, we regard as a sub-acute retinitis, due to glycosuria. In referring to authorities in relation to this disease, we do not find in those at our command a great number of examples of this affection. Jaeger* reports a case in which retinitis, mainly about the disc and of the papilla, with a few peripheral exudative spots, and with obscuration of the retinal vessels, was observed. Galezowski† says he has seen inflammations of the retina, iris, choroid, atrophy of the optic nerve, paralysis of the

*Atlas D'Ophthal., Pl. xiii, Fig. 64, and p. 96.

†Receueil D'Ophth., Feb. 1879, p. 75.

sixth nerve, hemiopia and amblyopia, due to glycosuria (as well as cataract). Schweigger does not speak of diabetic retinitis, but mentions, in *retinitis septica*, that the anatomical changes consist in hemorrhages and small white spots formed by thickened nerve fibres and fatty degenerated connective tissue corpuscles.* Zander† quotes Liebreich, who holds that “idiopathic affections of the retina are extremely rare,” and “to be almost always due to constitutional dyscrasia or blood poisoning.” Zander himself, speaking of the chronic inflammation and fatty metamorphosis which attend upon Bright’s disease, says:‡ “But it should be remarked that a similar or identical disease of the retina is seen in connection with diabetes mellitus, hippuria, benzuria, oxaluria, and so forth, as well as partially during pregnancy and lactation, and in syphilis.”

Stellwag§ says, while considering the causes of nephritic retinitis, “Similar ophthalmoscopic appearances have been observed in diabetes, and even in neuro-retinitis descendens, consequent upon cerebral affections.” The editor maintains that similar ophthalmoscopic appearances may be induced by any dyscrasia.

Wells|| says: “Retinitis is far more frequently due to some constitutional affection, or consequent on some other disease of the eye, *e. g.*, choroiditis.” * * * “It may also be caused by syphilis, by certain affections of the kidneys, especially Bright’s disease and diabetes, and by cerebral diseases.”

*Handbook of Ophth., 3d Germ. ed. by Fasley, p. 462.

†“The Ophth., Its Varieties and Use,” Carter. p. 156.

‡Ibid, p. 156.

§Treat. on the Dis. of the Eye, Am. ed., 4th rev., p. 190.

Treat. Dis. Eye, 3d ed., p. 373.

Walton,* speaking of hemorrhagic retinitis, says: "This has been described as occurring in diabetes, but not sufficiently authentic for me to accept the report."

Galezowski† says, as to the pathology of this form of retinitis: "What is the intimate cause of the alterations of the retina and optic nerve? The experiments of Claude Bernard have proved that wounds of the floor of the fourth ventricle occasion glycosuria. But if, on the one hand, the alterations of this region induce glycosuria, on the other hand it is not doubtful that the impoverishment of the blood occasions in its turn a variety of alterations in different parts of the body. We know, indeed, that purpura hemorrhagica (Trousseau), spontaneous gangrene (Marchal de Calvi), the softening, gray and red, in the fourth ventricle, and in the superior processes of the cerebellum, may be developed as a result of glycosuria."

"The alterations in the retina prove to us that it is really in the morbid state of the arteries that we should look for the proximate cause of the retinitis. As a result of the diseased state of the blood, and the affection of the fourth ventricle, transmitted through the vaso-motor system of nerves, the walls of the vessels do not perform their functions well, and the capillaries are transformed little by little, and subjected to fatty degeneration."

"In atrophy of the papilla, particularly in the brain, the material lesion is manifested, as has been observed by Luys, Becquerel, Trousseau and others; from thence it successively invades the optical centers and pro-

*Practical Treat. on the Dis. of the Eyes, 3d ed., p. 1107.

†Traite Des Mal. Des Yeux, 2d ed., p. 645.

duces their atrophy." He recites, at page 642 *ibid*, a case in his own practice, in which there was atrophy of the papilla, diminution in the size of its vessels, its outlines well marked, and the retina, in the region of the disc, not at all clouded and without trace of infiltration, similar to that found in albuminuric retinitis. There were also several hemorrhagic effusions at different points of the retina. He reports also another case seen by himself, as well as by Courtois, in which there was present only hemorrhagic extravasations of this tunic.

Mollein* has reported a case in which there was hemorrhagic spots and white exudations upon the retinae, and which recovered from diabetes.

Noyes† has recorded a case of this disease in which he has observed white exudations, more especially in the region of the macula, and similar in all respects to those which we detect in albuminuria. The optic nerves were congested without any clouding of the contours.

Boucher‡ reports a case of this affection in which there was venous hemorrhage in the retina in the region of the macula, and in the same work is cited the case of Mesnard, in which there was produced at once a glycosuric retinitis in both eyes, and about the same time one eye was seized with hemorrhagic glaucoma. * * * * *

According to Galezowski, the first two cases of this malady were reported by Desmarres.

**Ibid*, p. 642.

†*Trans. Am. Oph. Society*, 1869.

‡*Trait. Mal. Des Yeux. Gal.*, p. 643.

Roberts,* while not referring to diabetic cataract, cites Bouchardt's statement, that a fifth of diabetic cases are complicated with amblyopia. The author states that this effect may be temporary, and often finally ends in permanent blindness, which result, he says, seems to be caused by atrophy of the retina.

Pagenstecher and Gerth are silent in their splendid work as to glycosuric retinitis.† We find in it, however, delineations of morbid states of the retina due to albuminuria, some of them quite similar to those sometimes found in cases of glycosuric retinitis.

REMARKS.

The diabetes was cured in this case. I learned after several months that the little patient was still doing as well as when I last examined her.

From a consideration of the views of the above authorities I cannot believe this inflammation is a common affection. Whatever may be the nature of the diabetic process, we have ample evidence that all parts of the retina may be affected, though not simultaneously, or to an equal extent. In general, the disc and region about the papilla seems to suffer the most from the effects of the disease and the lesions would, in general appear to be less profound in this than in the albuminuric retinitis. It would seem that hemorrhages are more frequent in this than in the nephritic inflammation of this coat, in which latter disease exudations of plasma appear to be more commonly present. Still, the morbid process is essentially the same in

*Pract. Treat on Urinary and Renal Dis., 2d Am. Ed., p. 238

†Atlas of the Path. Anat. of the Eyeball.

both affections, viz., a lesion of nutrition, differing more in degree than in pathological characteristics.

It has not been positively shown that the disease begins primarily in the blood vessels of this tunic, and we know that the inroads of the affection do not always fall upon the same layers of this coat with equal intensity. Sometimes the effects tell mainly on the rods and cones, again on the nerve fibres, or possibly upon the ganglion elements. If the effusion be mainly serous, little damage may result to the nerve elements; if the extravasation be plastic, induration, distortion and destruction of these, with atrophy, may obtain, leaving the membrane impaired or unfit for vision. Proliferation and fatty degeneration form a part of the diseased process, and the small white spots mentioned seem to be due to these effects. Pigmentation and hemorrhagic extravasation may or may not be present as a result of this dyscrasia.

Neither is the disease always manifested with inflammatory symptoms, and yet profound lesions of nutrition seem to follow this affection of the retina—a membrane sparsely supplied with blood vessels, from the very nature of its functions. Can it, therefore, be that the lesions we are considering result from affections of the blood vessels alone, or even uniformly from the inflammatory process? May we not encounter here, as elsewhere, in the body, affections of nutrition without inflammation? What role does the vaso-motor system play, and what is the status of the sensory nerves in this affection?

While then we neither adopt the views of Virchow or those of Cohnheim, we must claim inflammation to be an affection of

both vessels and the perivascular area, as well as of the vaso-motor and sensory nerves.

I may add, in relation to this case, that my notes contain no mention of counter-irritation employed in the treatment. I cannot remember distinctly, but I think vesicants were employed. Further, there were no evidences of syphilis in the case.

Finally, to diagnose this disease it will not suffice to inspect the retina alone, but the urine must be also carefully tested and microscopically examined. If to the urine there be added a slight excess of the pure subacetate of lead, then filtered, and to the filtrate there be added a slight excess of *pure* sodium carbonate, to neutralize any excess of the lead salt (the process of M. Jolly*), Trommers, Prouts or Fehling's tests will act more satisfactorily.

I do not believe that the quantity of the sugar in the urine will always indicate the extent of the lesion of the retina, for do we not have glycosuric amblyopia without detectable lesions of the retina, and how seldom is this disease present in diabetes where a large quantity of sugar is voided in the urine.

*Cin. Lancet and Clinic, Feb. 7, 1880, from La France Med.